#### International Journal of Health Research, June 2011; 4(2): 75-82

© Poracom Academic Publishers. All rights reserved.

Available at http://www.ijhr.org

# **Original Research Article**

**Open** Access Online Journal

## Lipid and Lipoprotein Levels in Type 2 Diabetes Patients Attending the Central Regional Hospital in the Cape Coast Metropolis of Ghana

## Abstract

**Purpose:** To ascertain the prevalence of dyslipidaemia in diabetics in Cape Coast.

**Methods:** This preliminary outpatient-based cross-sectional study was conducted in 79 diabetic patients (22 males and 57 females) receiving treatment at the Central Regional Hospital (CRH) in the Cape Coast Metropolis. Serum lipid profiles of the fasting diabetic patients were determined between September, 2008 and May, 2009. In addition, demographic information, height and weight were measured and BMI was computed.

**Results:** BMI was significantly (P = 0.001) higher in females than in males, but the mean values of all the remaining measured parameters were comparable (P>0.05) between the sexes. No significant (P>0.05) correlation was observed between age or BMI and cholesterol levels. Percentage dyslipidaemia ranged from 7.60% to 55.70% in the study sample. A significantly (P<0.05) higher proportion of females than males in the entire sample were overweight/obese, had higher levels of total cholesterol (TCHOL) and TRG. No significant difference (P>0.05) was observed between proportions of individuals in both sexes who exhibited higher LDL-c but lower levels of HDL-c..

**Conclusion:** Lipid profile should be a routine test for all diabetics receiving treatment at the CRH to identify those at increased cardiovascular risk for immediate attention.

**Keywords:** Dyslipidaemia, Cardiovascular, Cholesterol, Body mass index

# Samuel Acquah<sup>1</sup> Johnson N Boampong<sup>2</sup> Justice Adusu<sup>3</sup> Emmanuel K Achampong<sup>4</sup> Jacob Setorglo<sup>1</sup> Dorcas Obiri-Yeboah<sup>5</sup>

<sup>2</sup>Department of Human Biology, School of Biological Sciences, UCC.

<sup>3</sup>Department of Laboratory Technology, School of Physical Sciences, UCC.

<sup>4</sup>Department of Medical Education School of Medical Sciences, UCC.

<sup>5</sup>Department of Microbiology, School of Medical Sciences, UCC.

#### \*For correspondence

Tel: +233(0)242341428

Email: s.acquah@uccsms.edu.gh

This article is available in Embase, Index Corpenicus, Scopus, PubsHub, Chemical Abstracts, Socolar, EBSCO, African Journal Online, African Index Medicus, Open-J-Gate, Directory of Open Access Journals (DOAJ) databases

Acquah et al

## Introduction

Diabetes mellitus is a metabolic disorder resulting from various hereditary and environmental factors [1]. It is characterized by high blood glucose levels [1]. Type 2 diabetes remains the most predominant form of diabetes in both developed and developing countries [2]. It is associated with insulin resistance, obesity, hypertension and other cardiovascular risk factors [3, 4].

Biochemically, a major cardiovascular risk factor in type 2 diabetes mellitus is dyslipidaemia associated with insulin resistance [5]. This pattern of dyslipidaemia, which transcends race and geographical location, is characterized by decreased levels of atheroprotective HDL-c, increased TRG but normal level of LDL-c [6,8]. Qualitatively, this pattern of lipid profile abnormalities is similar to that observed in HIV patients as a result of prolonged use of the highly active anti-retroviral drugs, especially, protease inhibitors [7]. The different patterns of dyslipidaemia in type 2 diabetics may result from varied underlying causes of the disease.

A spectacular global increase in obesity and diabetes, suitably called diabesity was predicted almost a decade ago [2]. Since then, the predicted diabesity trend has been observed in various countries [8]. In sub-Saharan Africa, including Ghana, the estimated burden of diabesity is expected to be greater than that for developed countries [9]. In spite of the expected considerable economic, social and health impact of diabetes in Ghana, country-specific data on prevalence of the condition are limited. In addition, the routine laboratory test requested by physicians for most diabetics is the fasting blood glucose but not lipid profile. This is because the major treatment goal for such patients is the attainment of normoglycemia with little attention for normolipidaemia, except for diabetic patients with established hypertension.

Abnormalities in lipid and lipoprotein levels in diabetics may occur with or without hypertension [1, 8]. These abnormalities, even in diabetics without hypertension, could pose equally significant morbidity and mortality risks to patients [5]. This brings to the fore, the need for information on lipid levels of the diabetic population in Ghana to ascertain the extent to which the abnormalities observed elsewhere exist in the Ghanaian setting. Such information may have implications on diabetes management to help improve the quality of life of diabetics in Ghana. For this reason, we designed this crosssectional study to measure the lipid profile of 79 type 2 diabetic patients attending the diabetes clinic of the Central Regional Hospital (CRH) in Cape Coast. Specifically, we measured total cholesterol (TCHOL), TRG, LDL-c and HDL-c to determine the prevalence of dyslipidaemia and the associated cardiovascular risk in our sample.

## Methods

#### Study area

The study was carried out in Cape Coast, the capital of the Central Region of Ghana. Cape Coast is situated 165km west of Accra on the Gulf of Guinea. According to the year 2000 Population and Housing Census, the population of the Cape Coast Metropolis stood at 82,291 with annual growth rate of 2.1% [10]. This gives an estimated population of 99,217 for the metropolis in 2009. The Metropolis covers an area of 122 square kilometers and is the smallest metropolis in the country. The Cape Coast Metropolis is located in the littoral anomalous zone of Ghana, making it experience high temperatures year round. The hottest months are February and March, just before the main rainy season, while the coolest months are between June and August. The annual rainfall range from 750 mm to 1000 mm. Cape Coast was chosen because it houses the CRH, the largest referral hospital in the region where diabetic patients in the region are referred.

#### Study design, patients and sample collection

This preliminary study was an out-patient-based cross-sectional study involving 22 male and 57 female diabetic patients attending CRH between

Acquah et al

September 2008 and May 2009. Inclusion criteria were confirmed type 2 diabetes and willingness to take part in the study. All patients included in the study were advised to fast for at least 12 hours prior to sample collection. Semi-structured questionnaires were administered to collect demographic and socio-economic information. Body weight (nearest 0.1 kg) and height (nearest 0.1 cm) were measured and the body mass index (BMI) was calculated as the ratio of weight in kilogrammes to the square of the height in meters.

Lipid analysis was performed on sera collected after 12 hours of fasting using the ATAC 8000 Random Access Chemistry System (Elan Diagnostics, Smithfield, RI, USA). The reagents used in the analysis were made by the same manufacturer. LDL-c was calculated according to the Friedewald equation: LDL-c = TCHOL -HDL-c – (TRG/2.19) [11]. Dyslipidaemia was defined as serum total cholesterol >5.2 mmol/L; serum LDL >2.6 mmol/L; serum triglycerides >1.7 mmol/L; and serum HDL <1.03 mmol/L [12]. Overweight/obesity was defined as BMI >25 kg/m<sup>2</sup> [11]. Cardiovascular risks were estimated by calculating the ratio of LDL-c, HDL-c and TCHOL [12]

#### **Ethical Approval**

The study was approved by the hospital authority. Informed consent was obtained from all the study patients. All protocols followed were in line with the ethical standards of the Ministry of Health, Ghana.

#### Statistical analysis

Data obtained were analyzed with the SPSS version 19.0 statistical software. Values were reported as mean ± standard deviation (sd) and percentages where appropriate. Mean values between the sexes were compared with one-way analysis of variance (ANOVA). Chi-square test was used to compare proportional data while Pearson correlation was used to assess the existence of linear association among the measured lipid parameters. A p-value of <0.05 was considered statistically significant.

#### **Results**

The mean age of the males did not differ significantly from that of the females but the females had significantly (P < 0.001) higher mean BMI than the males (Table 1). The mean levels of the various components of lipid profile did not differ (P>0.05) between the sexes. However, a significantly (P <0.05) higher proportion of females than males were overweight/obese  $(54.43\% \text{ vs } 12.66\%; \chi^2 = 14.75; P < 0.001)$ , had higher levels of TCHOL (31.7% vs 6.3%;  $\chi^2_=$ 4.57; P = 0.033) and TRG (22.79 vs 8.86;  $\chi^2$ =10.646; P =0.001). Comparable proportions of males and females (P> 0.05) had higher level of LDL-c (54.54% vs 61.40%;  $\chi^2 = 2.965$ ; P = 0.085) but lower level of HDL-c (13.63% vs 5.26%;  $\chi^2 = 2.972$ ; P = 0.09) (Figure 1).



Figure 1: Percentage overweight/obesity and dyslipidaemia according to gender

The calculated artheriogenic indices for the components of lipid profile are shown in Table 2). As indicated, the indices did not differ significantly (P>0.05) between the males and females. As expected, BMI correlated positively with weight (r = 0.74; p<0.001) but negatively with height (r = -0.55; P < 0.001) for the entire sample (Table 3). A significant positive correlation was observed between BMI and TRG levels in the females (r = 0.289; P = 0.03) but not in the males (r = 0.093; P = 0.681) or the entire sample (r = 0.162; P = 0.154). However, the levels of TRG correlated negatively with HDL-c in the entire sample (r = -0.235; P = 0.037). In addition, height correlated positively with HDL-c in males

	Mean ± S.D	95% C.I	Range	Ref. range	P-value
Age					
Male	$60.00 \pm 9.12$	59.62 - 63.81	44 - 81	-	0.134
Female	$55.63 \pm 12.28$	52.44 - 58.82	27 - 90	-	
BMI					
Male	$24.46 \pm 3.51$	22.99-25.93	18.96 - 29.79	18.50 - 24.90	0.001*
Female	$30.00 \pm 6.87$	28.22-31.78	20.96 - 30.00	18.50 - 24.90	
TCHOL					
Male	$4.74 \pm 0.91$	4.36-5.12	3.23 - 6.71	≤5.2	0.098
Female	$5.23 \pm 1.28$	4.90-5.56	1.99 - 8.55	≤5.2	
TRG					
Male	$1.68 \pm 1.04$	1.24-2.12	0.62 - 4.42	≤1.7	0.673
Female	$1.59 \pm 0.78$	1.39-1.79	0.43 - 4.85	≤1.7	
LDL-c					
Male	$2.71 \pm 0.96$	2.31-3.11	0.87 - 4.44	$\leq 2.6$	0.253
Female	$3.05 \pm 1.24$	2.73 - 3.37	0.76 - 7.03	≤2.6	
HDL-c					
Male	$1.46 \pm 0.45$	1.27 - 1.65	0.37 - 2.4	$\geq 1.03$	0.238
Female	$1.60 \pm 0.49$	1.47 – 1.73	0.65 - 2.76	≥ 1.03	

Table 1: Age, BMI and lipid profile of participants by gender

LDL-c = low-density lipoprotein, HDL-c = high-density lipoprotein, TCHOL = total cholesterol, TRG triglycerides, S.D = standard deviation, C. I. = confidence interval, BMI = body mass index, ref. = reference. P-value was computed by comparing mean values of the sexes. \* = P-value is significant.

$\begin{array}{c c c c c c c c c c c c c c c c c c c $							
$\begin{array}{c c c c c c c c c c c c c c c c c c c $	Variable	Mean ± S.D	95% C.I	Range	P-value		
$\begin{array}{cccccccccccccccccccccccccccccccccccc$	LDL-c/HDL-c						
$\begin{array}{cccccccccccccccccccccccccccccccccccc$	Male	$2.28 \pm 0.41$	2.11 - 2.45	0.69 - 9.78	0.573		
$\begin{array}{c c c c c c c c c c c c c c c c c c c $	Female	$2.08 \pm 0.15$	2.04 - 2.12	0.65 - 6.89			
$\begin{array}{cccccccccccccccccccccccccccccccccccc$	HDL-c/LDL-c						
$\begin{array}{cccccccccccccccccccccccccccccccccccc$	Male	$0.63 \pm 0.33$	0.49 - 7.7	0.10 - 1.45	0.906		
$\begin{array}{c c c c c c c c c c c c c c c c c c c $	Female	$0.62 \pm 0.33$	0.53 - 7.1	0.15 - 1.5			
$ \begin{array}{cccccccccccccccccccccccccccccccccccc$	TCHOL/HDL-c						
$\begin{array}{c ccccccccccccccccccccccccccccccccccc$	Male	$11.18 \pm 10.13$	6.95 - 15.41	2.80-49.21	0.839		
$\begin{array}{c c c c c c c c c c c c c c c c c c c $	Female	$11.66 \pm 9.30$	9.25 - 14.07	2.0-58.93			
$ \begin{array}{cccccccccccccccccccccccccccccccccccc$	HDL-c/TCHOL						
$\begin{array}{cccccccccccccccccccccccccccccccccccc$	Male	$0.32 \pm 0.10$	0.28 - 0.35	0.07 - 0.50	0.752		
$\begin{array}{c c c c c c c c c c c c c c c c c c c $	Female	$0.33 \pm 0.15$	0.29 - 0.36	0.12 - 1.13			
$ \begin{array}{cccccccccccccccccccccccccccccccccccc$	LDL-c/TCHOL						
$\begin{array}{cccccccccccccccccccccccccccccccccccc$	Male	$0.61 \pm 0.14$	0.55 - 0.67	0.39 - 1.01	0.291		
$\begin{array}{c c c c c c c c c c c c c c c c c c c $	Female	$0.56 \pm 0.18$	0.51 - 0.61	0.12 - 1.19			
$ \begin{array}{cccccccccccccccccccccccccccccccccccc$	TCHOL/LDL-c						
Female $2.02 \pm 1.02$ $1.75 - 2.29$ $0.84 - 8.09$ TRG/HDL-cNale $1.09 \pm 0.8$ $0.76 - 1.42$ $0.34 - 3.08$ $0.481$ Female $1.29 \pm 1.19$ $0.98 - 1.60$ $0.33 - 6.16$	Male	$1.73 \pm 0.40$	1.71 - 1.75	0.99 - 2.55	0.209		
TRG/HDL-c $Male$ $1.09 \pm 0.8$ $0.76 - 1.42$ $0.34 - 3.08$ $0.481$ Female $1.29 \pm 1.19$ $0.98 - 1.60$ $0.33 - 6.16$	Female	$2.02 \pm 1.02$	1.75 - 2.29	0.84 - 8.09			
Male $1.09 \pm 0.8$ $0.76 - 1.42$ $0.34 - 3.08$ $0.481$ Female $1.29 \pm 1.19$ $0.98 - 1.60$ $0.33 - 6.16$	TRG/HDL-c						
Female $1.29 \pm 1.19$ $0.98 - 1.60$ $0.33 - 6.16$	Male	$1.09 \pm 0.8$	0.76 - 1.42	0.34 - 3.08	0.481		
	Female	$1.29 \pm 1.19$	0.98 -1.60	0.33 - 6.16			

Table 2: Artheriogenic indices by gender

LDL-c = low-density lipoprotein, HDL-c = high-density lipoprotein, TCHOL = total cholesterol, TRG triglycerides, S.D = standard deviation, C. I. = confidence interval, BMI = body mass index, ref. = reference. P-value was computed by comparing mean values of the sexes. \* = P-value is significant.

(r = 0.443; P = 0.039) but not in females (r= 0.069; P = 0.61) or the entire sample (r = -0.033; P = 0.77). A significant positive correlation was found between TCHOL and LDL-c in the entire sample (r = 0.774; P < 0.001) and in both sexes (P

< 0.001). No significant correlation was observed between TCHOL and HDL-c in either the sexes or the entire sample (P>0.05).

Table 3: Correlations among measured parameters

	Correlation	P-value
	coefficient r	
DM INCL.		
BMI and Weight		
Entire sample	0.74	<0.001*
Male	0.86	< 0.001*
Female	0.778	< 0.001*
BMI and Height		
Entire sample	-0.55	< 0.001*
Male	0.156	0.487
Female	-0.551	< 0.001*
BMI and TRG		
Entire sample	0.162	0.154
Males	0.093	0.681
Females	0.287	0.03*
Height and HDL-c		
Entire sample	-0.033	0.77
Males	0.443	0.039*
Females	0.069	0.61
TCHOL and LDL-c		
Entire sample	0.743	< 0.001*
Male	0.693	< 0.001*
Female	0.747	< 0.001*
TRG and HDL-c		
Entire sample	-0.235	0.037*
Male	-0.295	0.182
Female	-0.206	0.125

LDL-c = low-density lipoprotein, HDL-c = high-density lipoprotein, TCHOL = total cholesterol, TRG triglycerides, BMI = body mass index, \* = P-value is significant.

## Discussion

The most common lipid and lipoprotein disorders found in patients with type 2 diabetes are high serum TRG and lower HDL-c levels [1,8]. In this study, higher levels of LDL-c and TCHOL were rather predominant. The deviation from the expected trend could be attributed to possible differences in adherence to treatment regimens and duration of disease condition of respondents in our study compared with previous ones [1,8].

The observed mean TCHOL level (4.74 mmol & 5.23 mmol for males and females respectively) was slightly lower than that observed by previous studies in Nigeria [1, 13] and diabetics with microvascular and macrovascular complications in Ghana [14] but higher than diabetics without complications [14]. The difference could be attributed to variations in characteristics of study participants in relation to age, duration of the diabetic condition, compliance with prescribed treatment regimen and dietary and lifestyle

factors [15]. Indeed, participants in the present study were older than those in previous studies [1, 14]. On the other hand, the relatively higher level of TCHOL found in the present study compared to diabetics without complications [14] may reflect the presence of micro- and macrovascular complications.

Hypercholesterolemia is considered as a risk factor for coronary heart disease and the ratio of TCHOL to HDL-c is considered the best lipid index for predicting cardiovascular event in various studies [15]. The observed mean level was higher compared with those reported earlier [1, 14]. The relatively higher ratio suggests increased risk of cardiovascular disease in the study group irrespective of the normal levels of HDL-c. There is a long-held view that high HDLc is cardioprotective with intensive efforts being made to increase HDL-c levels to positively affect patients with atherosclerotic heart disease and reduce the risk of those with increased risk of atherosclerosis [16]. Recent evidence, however, suggests that raising HDL-c levels per se may not result in decreased risk of cardiovascular disease [17] but rather the functional quality of HDL-c should be of major concern [17]. Moreover, the functional quality of HDL-c in diabetics has been found to be lower than that of healthy controls as HDL-c of diabetics is unable to reverse-transport cholesterol, has impaired anti-inflammatory and anti-oxidative properties and may even be proarteriogenic [18]. As such, the seeming normal levels of HDL-c found in the present study may not imply a reduced risk of cardiovascular incidence.

Serum LDL-c, viewed as the most atherogenic lipoprotein, has been found to range from normal to high levels in diabetics [15]. In a crosssectional study in the general population of Kumasi, Ghana, Eghan and Acheampong [11] found comparable levels of LDL-c between healthy diabetics and controls. Similar observations have been made by other researchers [6]. As expected in this study, the observed mean level of LDL-c was higher than the upper reference value, suggesting that respondents were at increased risk of cardiovascular attack. However, the observed LDL-c level was lower than the levels found in diabetics by previous

studies in Ghana [11, 14] and Nigeria [1, 13]. This finding may be ascribed to stricter adherence of respondents in the present study to treatment regimen than those of previous works [1, 11-14].

High level of TRG is one of the most common abnormalities in type 2 diabetes [6,13]. In the present study, the mean levels of TRG for both sexes fell within normal limit although about 28% of respondents had hypertriglyceridemia. Unlike previous studies [19, 20] which found positive correlation between TRG and LDL-c, the present study, rather observed TRG correlating negatively with HDL-c in the entire sample but positively with BMI in female respondents only. The negative correlation between HDL-c and TRG has been reported elsewhere [19] and may be related to the action of cholesteryl ester transfer protein (CETP), an enzyme which catalyses the transfer of cholesteryl ester (CE) and triglycerides between HDL-c and triglyceride-rich lipoproteins such as very lowdensity lipoprotein (VLDL) [21]. As the level of VLDL rises, more triglyceride from VLDL is exchanged for CE from HDL-c under the influence of CETP. The resultant triglyceride-rich HDL-c becomes more susceptible to hepatic lipase-dependent degradation and subsequent clearance from plasma [22]. As such. hypertriglyceridemia, with time, should lead to decreased level of HDL-c. In this study, normal levels of both TRG and HDL-c were observed, possibly due to treatment effect and/or reduced duration of diabetic condition. Nonetheless, approximately 28% and 8% of respondents suffered hypertriglyceridemia and low level of HDL-c respectively.

The observed positive correlation between height and HDL-c levels in male respondents but not in females or the entire sample requires further investigation involving a large sample to ascertain its medical relevance.

An association of obesity with diabetes has long been established [8]. The observed positive correlation between BMI and TRG in females agrees with the findings of previous studies [23, 24]. Indeed, the females in the current study had higher BMI than the male respondents and were either overweight or obese. Recently, Taskinen *et*  *al.* [25] established that increased triglycerides in obese men was due to increased secretion and severely impaired clearance of triglyceride-rich VLDL. Although the study of Taskinen *et al.* [25] did not include obese females, it might be possible that a similar mechanism might have been at play with respect to overweight or obese females. Hence, the normal triglyceride level of males in the present study could be ascribed to their normal BMI, an indication that the men had normal metabolism of triglyceride-rich VLDL.

## Conclusion

The study has unveiled the presence of dyslipidaemia in the diabetic population of the Cape Coast Metropolis. The observed prevalence of dyslipidaemia ranged from 8% to 56%. As a result, health professionals involved in the management of diabetes in the Cape Coast Metropolis need to pay attention to the lipid levels instead of the current focus on only normoglycemia. Lipid profile should be a routine test for diabetics in the Cape Coast Metropolis and indeed in Ghana.

## Acknowledgements

The authors would like to express their sincere gratitude to the management and staff of the Central Regional Hospital, Cape Coast and all the respondents who participated in this study for their support. We are also thankful to Prof. Magdalena Eriksson, Department of Biochemistry, UCCSMS and Dr. Isaac Galyuon, Molecular Department of Biology and Biotechnology, School of Biological Sciences, UCC, for reviewing the final manuscript.

## **Authors' Contribution**

SA contributed to the conception, design, data analysis, interpretation and drafting of manuscript; JNB was involved in the conception, design and critical review of manuscript; JA was involved in acquisition of data; EKA contributed to data analysis and drafting of manuscript; JS participated in drafting of manuscript; DOY took part in data acquisition and interpretation of data. All authors read and approved the final manuscript for publication.

## **Conflict of Interest**

No conflict of interest associated with this work.

## References

- Bello-Sani F, Bakari AG Anumah FE. Dyslipidaemia in persons with type 2 diabetes mellitus in Kaduna, Nigeria. Int J Diabetes & Metabolism 2007;15:9-13.
- Zimmet P, Alberti KG, Shaw J. Global and societal implications of the diabetes epidemic. *Nature* 2001;414:782-787.
- Yusuf S., Hawken S. Ounpuu T. Dans A., Avezum F., Lanas M., McQueen A., Budaj P., Pais J., Varigos and Lisheng L.; INTERHEART Study Investigators. Effect of potentially modifiable risk factors associated with myocardial infarction in52 countries (the INTERHEART study): case-control study. *Lancet* 2004;364: 937–952.
- Rutter MK, Meigs JB, Sullivan LM, D'Agostino RB, Wilson PW. Insulin resistance, the metabolic syndrome, and incident cardiovascular events in the Framingham Offspring Study. *Diabetes* 2005;54:3252–3257.
- Meigs JB, Wilson PW, Nathan DM, D'Agostino RBS, Williams K, Haffner SM. Prevalence and characteristics of the metabolic syndrome in the San Antonio Heart and Framingham Offspring Studies. *Diabetes* 2003;52:2160-2167.
- Motala AA, Pirie FJ, Gouws E, Amod A Omar MA. High incidence of type 2 diabetes mellitus in South African Indians: a 10-year follow-up study. *Diabet Med* 2003;20:23-30.
- Tershakovec AM, Frank I, Rader D. HIV-related lipodystrophy and related factors. *Atherosclerosis* 2004;174:1-10.
- Escobedo J, Schargrodsky H, Champagne B, Silva H, Boissonnet CP, Vinueza R, Torres M, Hernandez R, Wilson E. Prevalence of the Metabolic Syndrome in Latin America and its association with sub-clinical carotid atherosclerosis: the CARMELA cross sectional study. *Cardiovascular Diabetol* 2009; doi:10.1186/1475-2840-8-52.
- Danquah I, Bedu-Addo G, Mockenhaupt FP. Type 2 diabetes mellitus and increased risk for malaria infection. *Emerging Infectious Diseases* 2010; 16(10):1601-1604.
- Ministry of Local Government and Rural Development (MLGRD) [Updated 2006; cited September, 1 2011]. Demographic characteristics of Central Region. Available from: http://www.ghanadis tricts.com.
- Eghan Jr, BA, Acheampong JW. Dyslipidemia in Outpatients at General Hospital in Kumasi, Ghana: Cross-sectional study. *Croatian Medical Journal* 2003; 44(5):576-578.

- Warnick G. R., Myers G. L., Cooper G. R. and Rifai N. (2002). Impact of the third cholesterol report from the adult treatment panel of the national cholesterol education program on the clinical laboratory. *Clin Chem* 48:11-17.
- Ogbe PJ, Digban KA, Idoko OA. Laboratory evaluation of dyslipidemia in patients with type 2 diabetes in Makurdi Metropolis. J Med Lab Sci 2011;2 (1): 54-57.
- Adinortey MB, Gyan BE, Adjimani J, Nyarko P, Sarpong C, Tsikata FY, Nyarko AK. Dyslipidaemia associated with type 2 diabetics with micro and macrovascular complications among Ghanaians. *Ind J Clin Biochem.* 2011;doi10.1007/s12291-010-0101-3.
- 15. Sert M, Morgul G, Tetiker BT. Diabetic dyslipidemia is a well-known issue, but what about lipoprotein a levels in Type 2 diabetics? *Int J Diabetes & Metab* 2010; 18:81-87.
- Kumar A. and Sivakanesan R. Serum lipid profile abnormality in predicting the risk of myocardial infarction in elderly normolipidaemic patients in South Asia: a case-controlled study. *Internet J Altern Med* 2009;6:2.
- deGoma EM, deGoma RI, Rader DJ. Beyond highdensity lipoprotein levels evaluating high-density lipoprotein function as influenced by novel therapeutic approaches. J Am Coll Cardiol 2008;51:2199-2211.
- Kastelein JJ, van Leuven SI, Burgess L, Evans GW, Kuivenhoven JA, Barter PJ, Revkin JH, Grobbee DE, Riley WA, Shear CL, Duggan WT, Bots ML. Effect of torceptrepid on carotid atherosclerosis in familial hypercholesterolemia. N Engl J Med 2007;356:1620 – 30.
- Watts GF, Barret PH, Chan DC. HDL metabolism in context: looking on the bright side. 2008;Curr Opin Lipidol 19:395-404.
- 20. Sorrentino SA, Besler C, Rohrer L, Meyer M, Heinrich K, Bahlmann FH, Mueller M, Horváth T, Doerries C, Heinemann M, Flemmer S, Markowski A, Manes C, Bahr MJ, Haller H, von Eckardstein A, Drexler H, Landmesser U. Endothelial-vasoprotective effects of high-density lipoprotein are impaired in patients with type 2 diabetes mellitus but are improved after extended-release niacin therapy. *Circulation* 2010; 121:1-4.
- Hayek T, Azrolan N, Verdery RB, Tova AW. Hypertriglyceridemia and cholesteryl ester transfer protein interact to dramatically alter high density lipoprotein levels, particle sizes, and metabolism. Studies in transgenic mice. J Clin Invest. 1993;92:1143–52.
- Horowitz BS, Goldberg IJ, Merab J, Vanni TM, Ramakrishnan R, Ginsberg HN. Increased plasma and renal clearance of an exchangeable pool of apolipoprotein A-I in subjects with low levels of high density lipoprotein cholesterol. J Clin Invest 1993;91:1743–52.
- 23. Dolevall A, Johnsson S, Wilhelmsen L, Rosengren A. Increased levels of triglycerides, BMI, BP and low physical activity increase the risk of diabetes in Swedish Women. A prospective 18-year follow-up of BEDA study. *Diabetic Med* 2004;21(6):615-22.

#### Acquah et al

- 24. Li J, Liao C, Su H, Peng Q, Zhhang Z, Yang Q, Yan S. Gender and BMI influence triglycerol level in oral fatty tolerant test in healthy young persons. *Lipids in Health and Disease* 2011;doi:10.1186/1476-511x-10-109.
  - 25. Taskinen MR, Adiels M, Westerbacka J, Sonderlund S, Kahri J, Lundbom N, Lundbom J,

Hakkarainen A, Olofsson S-O, Orho-Melader M Boren J. Dual metabolic defects are required to produce hypertriglyceridemia in obese subjects. *Throm Vasc Biol* 2011; doi:10.1161/ATVBAHAH. 111. 224808.